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Table 6 Comparisons of hemodynamics of uremic hypertensive group and hypertensive group ($\bar{x} \pm s$).

Groups	High volume		Hyperkinetic		High volume combined with hyperkinesis		High volume combined hyperkinesis and high resistance		High resistance	
	No.	%	No.	%	No.	%	No.	%	No.	%
Uremia	20	19.2	3	2.9	45	43.3	14	13.5	7	5.8
Hypertension	4	5.3	1	1.3	25	32.9	25	32.9	11	14.5
χ^2	7.821		0.497		1.989		9.771		3.890	
P value	0.005		0.481		0.158		0.002		0.049	

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Table 7 Logistic regression analysis of factors leading to uremic hypertension.

Factors	χ^2	*P* value	OR	95% confidence interval of OR
Age	14.804	0.000	1.063	0.931 ~ 1.195
CO	26.303	0.000	3.436	3.361 ~ 3.511
EF	10.658	0.001	1.409	1.225 ~ 1.593
LSV	4.904	0.027	1.012	0.612 ~ 1.412
VFC	11.644	0.001	3.235	3.067 ~ 3.403
CVP	21.473	0.000	1.306	1.215 ~ 1.397
MIM	14.232	0.000	1.005	0.867 ~ 1.143

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F-actin Rearrangement is Involved in Decreased Cell-surface Expression of Slo1 and BK_{Ca} Channel Inhibition Caused by Hypoxia in a Conditionally Immortalized Human Podocyte Cell Line

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There is much evidence to show that hypoxia and ischemia compromise the structure and function of podocytes. Chronic hypoxia involved in nephrosclerosis is an alternative mechanism for the pathogenesis of rapidly progressive glomerulonephritis. Large-conductance Ca^{2+} activated K^+ channels (BK_{Ca} channels) encoded by the Slo1 gene are characterized as mechanosensitive and oxygen-sensitive channels in podocytes. However, whether BK_{Ca} channels are involved in the podocyte response to chronic hypoxia and the possible underlying mechanisms remain unclear. A conditionally immortalized human podocyte cell line transfected with the temperature-sensitive SV40 gene construct was cultured and differentiated. We used the patch clamp technique to show that the exposure of human podocytes to 2% O_2 for 24 hours causes a significant reduction in BK_{Ca} channel currents. FITC-phalloidin staining display that hypoxia and cytochalasin D cause F-actin rearrangement and depolymerization in podocytes. Furthermore, cell-surface biotinylation assays indicate a marked decrease in surface expression of Slo1 in anoxia. However, the total amount of Slo1 expression in whole cell has no significant variation after hypoxic treatment. Thus, we conclude that chronic hypoxia inhibits podocytes BK_{Ca} channel currents by F-actin rearrangement and reduced Slo1 expression on cell membrane. These findings provide new insight into the mechanism underlying the cellular responses of podocytes to hypoxia.

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A Nationwide, Multicenter, Cross-sectional Study on Hypertension in CKD Patients

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